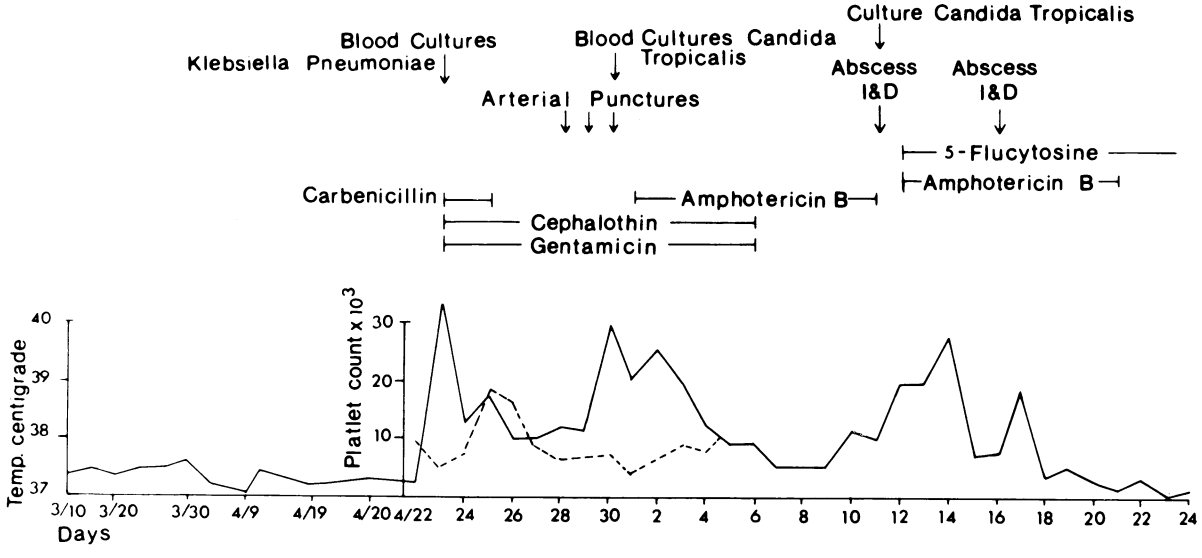
# Correspondence

*The Editorial Board will be pleased to receive and consider for publication correspondence containing information of interest to physicians or commenting on issues of the day. Letters ordinarily should not exceed 600 words, and must be typewritten, double-spaced and submitted in duplicate (the original typescript and one copy). Authors will be given an opportunity to review any substantial editing or abridgment before publication.*

## Candida tropicalis Abscess Following Arterial Puncture

TO THE EDITOR: Although arterial peripheral blood gas (ABG) determinations are routinely used in the management of complicated medical and surgical cases, and are rarely associated with problems, we wish to report a significant case of severe Candida tropicalis abscess formation secondary to arterial puncture.

A 25-year-old woman with acute lymphocytic leukemia was initially treated unsuccessfully with vincristine (2 mg per week for four weeks) and prednisone (110 mg per day). Remission occurred with a second course of treatment consisting of daunomycin (120 mg per day for three days), cytosine arabinoside (175 mg twice a day for nine days), thioguanine (175 mg twice a day for nine days) and prednisone (110 mg per day). Subsequently a small bowel obstruction with bloody diarrhea, a temperature of 40°C (104°F) and hypotension developed. Intravenous therapy with carbenicillin, cephalothin and gentamicin was initiated and the patient's fever abated in 48 hours; Klebsiella pneumoniae were obtained from cultured blood specimens. Fever recurred in 48 hours and multiple blood specimens grew pure cultures of Candida tropicalis even after removal of all indwelling venous catheters. The patient was treated with amphotericin B (136 mg per day for ten days). During the second febrile episode three ABG specimens were obtained, two from the patient's left radial artery and one from the right brachial artery; the patient was also leukopenic, thrombocytopenic (platelets, 5,000 to 8,000 per cu mm) and had an abnormal coagulation profile. Four days later the peripheral leukocyte count had recovered (leukocytes, 2,100 per $\mu$l, with 85 percent polymorphonuclear neutrophils) and at that time she complained of pain and swelling at the site of a previous puncture of the right brachial artery. During the following week a frank abscess developed at that site and upon incision 400 ml of purulent material was obtained, with yeast formations visible on microscopic examination




I&D = incision and drainage

**Figure 1.**—Clinical course of patient in whom Candida tropicalis abscess developed after arterial punctures.

and subsequently identified on culture as Candida tropicalis. A small abscess noted on the patient's left wrist at the site of another arterial puncture was also drained and the fluid produced no growth on culture. The patient was treated for ten days with a combination of amphotericin B and flucytosine, resulting in the resolution of fungemia and healing of the abscess cavities. The clinical course of this patient is shown in Figure 1.

Arterial blood gas determinations are frequently necessary in critically ill patients and usually do not cause complications. Petty and co-workers[1] reported 475 arterial punctures without complications in 54 patients. Fleming and Bowen[2] found in their series of more than 4,000 ABG determinations that hematoma formation was the most common complication with an incidence of only 0.58 percent, and that there were no serious sequelae from this mild complication. The risk of hematoma formation and its consequences appears to increase in patients receiving anticoagulant therapy. Neviaser and associates[3] reported that in 13 patients hematomas developed following femoral or brachial artery punctures while the patients were receiving therapeutic doses of heparin. Luce and co-workers[4] similarly reported that in seven patients receiving anticoagulant therapy compressive neuropathy developed secondary to hematoma formation from brachial artery punctures; they felt that the radial artery was the preferred site for ABG sampling in patients receiving anticoagulant therapy.

Infection of a hematoma is a recognized surgical complication and Neviaser[3] reported two cases in his series. He did not describe the results of Gram stains or cultures; however, he did note that primary therapy was incision and drainage. The occurrence of a Candida tropicalis abscess in our patient is unusual but not surprising. It has been shown that patients with hematologic malignancies or immune suppression have a greater risk of significant fungal infection. Also, hematoma infections with Candida species have been noted in surgical patients. We believe this is the first time that Candida tropicalis has been associated with an abscess following arterial puncture for ABG determinations.

This observation shows that even the simplest of invasive procedures can be associated with a risk. It should also be stressed that these risks are magnified in patients receiving anticoagulant therapy who have immunologic disorders secondary to their underlying illness or chemotherapy. To minimize the risk of hematoma formation and its sequelae, we recommend that ABG samples should not be obtained from brachial or femoral artery sites. We also feel that patients should be observed for evidence of abscess formations if arterial punctures are done during a period of septicemia.

JAMES F. HANLEY, MD
ALLAN F. AVBEL, MD, MS
ARTHUR R. KNODEL, MD
JOHN K. PODGORE, DO
*Infectious Disease Service*
*Madigan Army Medical Center*
*Tacoma, Washington*

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3. Neviaser RJ, Adams JP, May GI: Complications of arterial puncture in anti-coagulated patients. J Bone Joint Surg [Am] 58: 218-220, Mar 1976

4. Luce EA, Futrell JW, Wilgis EF, et al: Compression neuropathy following brachial arterial puncture in anti-coagulated patients. J Trauma 16:717-721, Sep 1976

## Hypertrophic Cardiomyopathies

TO THE EDITOR: We would like to comment on Dr. J. Michael Criley's interesting editorial remarks in the April issue (Criley JM: The bottom line syndrome—Hypertrophic cardiomyopathy [Editorial Comment] West J Med 130:350-353, Apr 1979) concerning our review of hypertrophic cardiomyopathy (Ross J Jr, Shabetai R, Curtis G, et al: Nonobstructive and obstructive hypertrophic cardiomyopathies—UCSD School of Medicine and San Diego VA Medical Center [Specialty Conference]. West J Med 130:325-349, Apr 1979), which appeared in the same issue. We acknowledge here, as we did there, his important contributions to the understanding of the pathophysiology of the hypertrophic cardiomyopathies.

It is hoped that we succeeded in making clear to our readers that we do not consider outflow tract obstruction to be the "bottom line" in hypertrophic cardiomyopathy. We emphasized that a systolic pressure gradient frequently is absent and when present is variable and unrelated to prognosis or symptoms; moreover, the mechanisms of its genesis and its abolition by myectomy are still not understood. Our preference for the term *hypertrophic cardiomyopathy* over the term hypertrophic subaortic stenosis indicates our belief that this is a disease of heart muscle, albeit mainly of the ventricular septum, and we agree that the disorder may create major disturbances in the diastolic as well as the systolic function of the heart. Thus, to state that left ventricular outflow tract